

Editorial Comment

Evaluation of Pulmonary Venous Flow by Transesophageal Echocardiography*

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Transesophageal echocardiography has offered cardiologists a new, clear window on the heart. With its proximity to the heart and its enhanced spatial resolution, the technique offers detail heretofore seen only by the cardiac pathologist. The use of Doppler ultrasound will widen applications of transesophageal echocardiography to intraoperative assessments of mitral valve area, interrogation of left ventricular outflow tract velocity to obtain postmyomectomy pressure gradients for hypertrophic cardiomyopathy and many other purposes.

The current study. Pulsed Doppler echocardiography allows the analysis of specific areas in the cardiac chambers, such as pulmonary venous inflow. In this issue of the Journal, Castello et al. (1) provide normal values of transesophageal echocardiography-derived pulmonary venous inflow. They state that the left upper pulmonary vein offers the most accessible data with the least spectral broadening. Furthermore, they point out that the pulmonary venous inflow velocity signal has four phases: 1) systolic inflow due to atrial relaxation; 2) flow due to ventricular contraction with the resultant "drop" of the mitral anulus toward the apex of the left ventricle; 3) diastolic inflow secondary to opening of the mitral valve; and 4) a negative signal secondary to atrial contraction. In transthoracic studies one rarely observes atrial contraction and biphasic ventricular systolic flow.

Currently, the pulmonary venous inflow velocity signal is utilized in the grading of mitral regurgitation (2,3). Flow profiles in the pulmonary veins that indicate systolic flow reversal may be as accurate as jet area in such grading. The magnitude and shape of the pulmonary venous inflow velocity signal are due to several factors. Castello et al. note its dependence on left atrial compliance, left atrial pressure, left ventricular compliance and the status of the mitral valve

(that is, whether it is stenotic, competent or regurgitant, and other hemodynamic factors that affect left ventricular filling dynamics, including heart rate, preload and incomplete or complete relaxation).

This descriptive report seeks to describe the normal findings of the pulmonary venous inflow velocity signal. One will understand abnormal findings when the range of normal is better characterized. Transthoracic echocardiography provided insufficient details of the quadriphasic nature of the pulmonary venous inflow velocity signal; often one could only see a biphasic signal. Furthermore, it appears that the absolute velocity measurements varied with the position of the probe in relation to the pulmonary vein orifice, with diastolic velocities tending to decrease as the sample volume was moved distally into the vein. This study establishes the standard for sample volume location and orientation that previous studies neglected.

Comparison with previous studies. One weakness of this study is that it neglected common hemodynamic factors that could influence measurement of the pulmonary venous inflow velocity signal. Specifically, we know that elevated heart rate decreases diastolic filling time and thus would shorten and decrease the amplitude of some of the components of the pulmonary venous inflow velocity signal. Furthermore, it would have been useful to determine how this signal correlated with the transthoracically determined mitral inflow, a more accessible measure of left ventricular diastolic function (4). Nishimura et al. (5) have previously published a comparison of pulmonary venous and mitral inflow velocities. They concluded that there was a direct correlation between changes in mitral E wave velocity and the early peak diastolic velocity in the pulmonary venous inflow velocity signal ($r = 0.61$). This study also revealed a strong correlation between the deceleration time of the mitral inflow and pulmonary venous inflow velocity signals in early diastole ($r = 0.8^*$). Their study was performed on patients undergoing coronary bypass graft surgery, and therefore data were obtained on pulmonary capillary wedge pressure, arterial pressure and cardiac output. An important question regarding the pulmonary venous inflow velocity signal is whether or not it has any relation to mean left atrial pressure. Nishimura et al. (5) determined that the degree of flow reversal with atrial contraction correlated highly with mean pulmonary capillary wedge pressure ($r = 0.81$).

Another study by Kuecherer et al. (6) that included transesophageal echocardiography and Swan-Ganz catheter measurements in patients undergoing cardiovascular surgery also addressed the relation between the pulmonary venous inflow velocity signal and pulmonary capillary wedge pressure. These investigations determined that Doppler variables of the pulmonary venous inflow velocity signal correlated more strongly with mean left atrial pressure than did Doppler variables of mitral inflow. The systolic fraction of the pulmonary venous inflow velocity signal (systolic velocity-time integral divided by the sum of systolic and early diastolic

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velocity-time integrals) correlated highly with mean left atrial pressure ($r = 0.88$). With respect to mitral inflow measurements, the ratio of mitral peak early diastolic flow velocity to pulmonary venous peak late diastolic flow velocity correlated with mean left atrial pressure ($r = 0.43$). A decrease in peak pulmonary venous inflow systolic velocity and systolic velocity-time integral should correlate strongly with mean left atrial pressure. As this pressure increases, the gradient from the pulmonary veins into the left atrium should decrease, leading to a decrease in peak systolic pulmonary venous inflow velocity.

Implications for assessing diastolic function. Pulmonary capillary wedge pressure has been used as an estimate of preload. The direct measure of preload is end-diastolic fiber length or its three-dimensional equivalent, end-diastolic volume. The latter is altered by end-diastolic pressure. Thus, it is not surprising that, under conditions of changing compliance such as ischemia, the pulmonary capillary wedge or left atrial pressure may not truly reflect end-diastolic volume (7). The Doppler-derived pulmonary venous inflow velocity signal may give us another window on changes in left atrial pressure, but it will not truly measure end-diastolic volume (preload) (8). Any single variable that attempts to indirectly assess end-diastolic volume (preload) will be inherently limited. This fact will need to be acknowledged in the application of the pulmonary venous inflow velocity signal as a measure of diastolic function.

Restrictive cardiomyopathy. There are some preliminary comparison data on the pulmonary venous inflow velocity signal and two conditions with abnormal diastolic function. Schiavone et al. (9) recently compared pulmonary venous inflow velocity signal data from patients with a form of restrictive cardiomyopathy (amyloid heart disease), or constrictive pericarditis with values in a normal control group. In normal subjects systolic flow velocity/diastolic flow velocity remained constant throughout the respiratory cycle. However, in restrictive cardiomyopathy, diastolic flow velocity was much greater than systolic flow velocity throughout the cycle. In patients with constrictive pericarditis, systolic flow velocity was greater during inspiration, and systolic and diastolic flow velocities were nearly equal during expiration. The investigators (9) postulate that the difference in the two conditions is due to a noncompliant left atrium in patients with amyloid heart disease. Atrial thickening is not unusual in this condition, and thus a stiff left atrium would lead to decreased systolic flow velocity accounting for the low systolic/diastolic flow velocity ratio in amyloid heart disease. The stiff left atrium would lead to higher left atrial pressure, limiting the systolic inflow into the left atrium, and decreasing systolic flow velocity.

Cardiac tamponade. Work by Appleton et al. (10) revealed the importance of mitral and tricuspid Doppler inflow assessment in the diagnosis of cardiac compression in cardiac tamponade. These studies illustrate the importance of major respiratory variation in the mitral and tricuspid Doppler inflow measurements in patients with cardiac tamponade

or constrictive pericarditis. Pulmonary venous inflow velocity signal assessment by transesophageal echocardiography may increase our understanding of the mechanisms of cardiac tamponade and pericardial disease in humans. This application may be important, in light of research by Fowler and Gabel (11) that the hemodynamic effects of surgically created tamponade are the result of compression of the atria (or the venae cavae) and the *pulmonary veins*, not the right ventricle. Therefore, as one measured the pulmonary venous inflow velocity signal in cardiac tamponade one would expect to see a marked decrease in peak diastolic and systolic velocities and the systolic velocity-time integral (measures of atrial filling). Further research is needed to explore the possible benefits of pulmonary venous inflow velocity signal measurements in the assessment of pericardial diseases and other states of abnormal diastolic function.

Conclusions. The study by Castello et al. (1) is important in that it delineates the normal four-phase pattern of the pulmonary venous inflow velocity signal. If we understand how the quadriphasic pulmonary venous inflow velocity signal changes under conditions of normal and elevated mean left atrial pressures, we may be able to use this technique to qualitatively or semiquantitatively estimate mean left atrial pressure. This ability may further assist us in the management of acutely ill patients who are undergoing transesophageal echocardiography without a Swan-Ganz catheter, because it offers information on left ventricular systolic and diastolic function. Current work by Lambertz et al. (12) that utilizes rapid atrial pacing to detect ischemic wall motion abnormalities during transesophageal echocardiography might use the pulmonary venous inflow velocity signal to detect diastolic dysfunction. Further combined hemodynamic and transesophageal echocardiographic studies will be required to delineate the effects of other factors that may influence the pulmonary venous inflow velocity signal.

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